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Contemporary approaches to the trigeminal neuralgia therapeutic management

Abstract: Trigeminal neuralgia is one of the most widespread cases of prosopalgia characterized by a high intensiveness of pain attacks as well as by an exclusive resistance to different therapeutic methods. [2, 39–41; 3, 326–329]. Specific paroxysmal character of pain attacks in trigeminal neuralgia defines its therapy techniques. The first medication which gave a significant effect in the therapy of trigeminal neuralgia was Dilantin. Blom in 1973 was the first who used anticonvulsants for pain management (Finlepsin, Carbamazepine).

Keywords: Trigeminal neuralgia, paroxysmal pain, treatment, anticonvulsants.

Purpose of research: to analyze contemporary methods of the trigeminal neuralgia treatment.

Materials and methods: analyze of contemporary literature concerning the basic approaches to the trigeminal neuralgia treatment.

Results of research: Specific paroxysmal character of pain attacks in trigeminal neuralgia defines its therapy techniques. The first medication which gave a significant effect in the therapy of trigeminal neuralgia was Dilantin. Positive results were achieved by a combination of Dilantin with Am-

inazin (L. G. Erochin, 1973). Blom in 1973 was the first who used anticonvulsants for pain management (Finlepsin, Carbamazepine).

The review of numerous researches about Carbamazepine makes clear the fact that 70–90% of patients had a significant positive effect after the first intake of this drug (B. A. Karlov and O. N. Savitskaya, 1980, V. A. Smirnov and others, 1968, Blom, 1963). However a long-term medication of trigeminal neuralgia by Carbamazepine leads to a gradual reduction of its analgesic effect owing to the drug tolerance elaboration (Hoppere R., 1980). That's why a NTN medications pharmacokinetic study is especially topical.

The main aim of treatment of trigeminal neuralgia is an eradication of a pain's reason (bad tooth, inflammation process in adjacent zones etc) and definition of a symptomatic therapy (pain management, restoration of the nerve's functions and structure).

According to the recommendations for the NTN pain control by the European Federation of Neurological Societies (2009) the first line medications are Carbamazepine in a dose of 200–1200 mg/day and Oxcarbamezepine 600–1800 mg/d. Example of a medication schema: 2 days a patient intakes ½ t (100 mg) 3 times a day, daily dose 300 mg; then 2 days 1 t (200 mg) 2 times a day (daily dose 400 mg); then 2 days 1 tablet (200 mg) 3 times a day (daily dose 600 mg). The effect must be felt within 48 hours. In the case of failure a dose can be increased up to 1200 mg. Medication by Carbamazepine should be started by 50 mg per day and increased up to 600–800 mg, and in 3–4 weeks daily dose must be significantly reduced to a maintenance dose. The medications of the second line are Lamotrigine (400 mg/day) and Baclofen (40–80 mg/day). There are researches describing the effectiveness of Phenytoin, Gabapentin, Clonazepam, Valproate. Such a therapy is especially effective with a classic (idiopathic) NTN [9, 30–32; 10, 306].

NSAIDs are frequently used for trigeminal neuralgia treatment: Xefocam (Lornoxicam) producing a well expressed analgesic and anti-inflammatory effect. Xefocam is especially effective with a NTN of peripheral genesis [10, 306].

Besides anticonvulsant, antidepressant and NSAID, neuro-metabolic medications, antioxidant, antihypoxants (Actovegin) can be also rationally administrated. Researches of several authors prove that usage of neuro-metabolic drugs favors normalization of metabolic processes in trigeminal nerve system, eradication of trigger points and prevention of pain attacks. B vitamins are also used for treatment of trigeminal nerve neuralgia; they produce a neurotropic action, analgesic effect, and nerve regeneration capacity. Maximal effect of B vitamins can be reached by an intake of multivitamin complexes (Neurobion). Its effectiveness is explained by a cumulative, antinociceptive and neurotropic action of all components. It favors with inhibition of nociceptive impulsation and with positive influence on axons and TN medullary sheath regeneration processes. Nerve reconstruction leads to a coherent passage of afferent impulses and normalization of cerebrospinal nucleus's gelatinous substance activation, which blocks a portal for a pain impulsation flow.

Physiotherapeutic procedures are also used for NTN treatment: electrophoresis, phonophoresis, amplimpulse on trigger zones, as well as laserotherapy.

In the case of failure of the above-listed measures a surgical operation can be recommended (Sano K. 1987) [8]. Nowadays the following operations are performed (E. I. Kandel, 1981):

1) Operation (transaction, decompression, electro stimulation) on trigeminal ganglion and sensory root of trigeminal nerve;

2) Transaction of a TN conduction tract and its sensor nuclei in medulla, at the level of thalamus and pain-conducting way from thalamus to cerebral cortex;

3) Operation (transaction, alcohol block) on the three branches of trigeminal nerve.

The most effective surgical method of trigeminal neuralgia treatment is percutaneous stereotaxic destruction of trigeminal ganglion elaborated by Sweet, Wepsic, (1978) Siegfried (1977). Chemical and hydrothermal rhizotomies are effective and are rarely followed by remissions. (L. Y. Livshits, 1965; A. A. Choudnovsky, 1983), but sometimes they provoke a total prolapse of trigeminal nerve functions and lesion of adjacent visceral-nervous units. For the elimination of these complications Sweet and Wepslo 1978, elaborated a method of selective destruction of pain-conducting fibers of a trigeminal nerve's root [10, 306].

Among the surgical methods a microvascular decompression proposed by P. Jannetta (1977) found a large application scope. The operation consists of trepanation of posterior cranial fossa, revision of interrelations between the TN root, upper cerebellar (rarely of the anterior inferior cerebellar) artery and superior petrosal vein. In the case of compression of the root by vessels the last ones must be separated and drawn aside. Synthetic pad is placed between the root and the vessels (Pand R. W., 1982; Pollaek I. F., 1988). The average effectiveness of such operation is 77,5%.

Despite the encouraging results of surgical methods, the problem of remissions exists. That's why elaboration of new methods of trigeminal neuralgia treatment and definition of strict surgery indications is still acute.

Afanassieva E. V. proposed a schema of conservative treatment of patients diagnosed with trigeminal neuralgia, who can't undergo the operation of microvascular decompression because of somatic contraindications. The treatment is aimed at the reduction of nociceptive impact of pulse strokes on the root. The schema is the following: blocking with Kenalog, Lidocaine and Vitamin B12, in the region of peripheral nidus of dymyelination, perineurally: with neuralgia of the third branch — near the oval foramen, in the case of neuralgia of the second branch — in the region of pterygopalatine fossa. 3–5 blockings every second day are necessary. Besides this, drugs for the remyelynation of the trigeminal nerve are administrated: Lipoic acid (Tioctacide, Berlitione), B-vitamins (Milgamma Neuromultiv-

it). For the increase of distance between the compressing artery and the root of trigeminal nerve at the expense of its volume — diuretics (Glycerin 0,5–0,8 g/kg) are prescribed under hematocrit guidance. So 37 patients were examined according to this schema. 27 of them 78% had a long-lasting remission. 8 of them had a pain of a very high intensiveness according to the visual analogue scale which was appreciated as a moderate pain after treatment. Remission of pain syndrome after the conservative therapy in 5 cases lead to a microvascular decompression in 3, 6, 18, 22, 24 months. Taking into account that the majority of patients suffering from trigeminal neuralgia are elderly persons — pathogenetic treatment is more relevant for them. In contrast to surgical interventions a conservative therapy excludes the development of deafferentation syndrome which are quite as much afflicting for them as trigeminal neuralgia [1, 24–26].

Researches of the authors (Karpov S. M., Christoforando D. Y., Baturin V. A., Karpov A. S. 2013) make a proposition that a pain syndrome in NTN is a consequence of autoimmune processes of trigeminal nerve structures, provoked by anamnestic catarrhal infections, as well as inflammation reactions after dental operations. These facts result in rising antibody titer to the myelin basic protein, inducing a process of demyelization. In this regard a therapy with Glucocorticoides (Metypred) was proposed to block the autoimmune aggression for an improvement of neurotrophic processes — Tyogamma, Neuromultivit. Research data confirmed that these drugs not only reduce pain sensations but also decrease the processes of demyelination and lead to a restoration of nerve structures resulting in a long-term remission [3, 326–329; 7, 16–19].

In the researches dedicated to botulinum toxin type A in treatment of trigeminal neuralgia undertaken in New York Center for Voice and Swallowing Disorders, St Luke's-Roosevelt Hospital, New York, U. S.A., Guardiani E., Sadoughi B, Blitzer A., Sirois D. (2013), in the most part of patients undergoing the intracutenous and subcutaneous infusions is shown its effectiveness. The most common side effect was facial nerve paresis. So the botulinum toxin type A injections offer a safe, effective local treatment of trigeminal neuralgia. This method can be an alternative one in patients with intolerance or irresponsiveness to pharmacological medication of the first line [16, 413–417.].

The researches undertaken by Arai YC, Hatakeyama N, M Nishihara, Ikeuchi M, Kurisuno M, Ikemoto T. studying trigeminal neuralgia demonstrate the effectiveness in a great part of patients of treatment by Carbamazepine, Gabapentine and Pregabaline separately or in combination. However several patients don't feel any improvement or suffer from pronounced.

Conclusion: The analysis of Russian and foreign literature brings us to the point that the trigeminal nerve neuralgia is provoked by reasons, mechanisms of occurrence and development of pain syndrome of different character. At the contemporary stage of neurology from scientific and from practical point of view the problem of trigeminal nerve neuralgia remains discussable and acute especially if considering the nondecreasing specific weight of the disease, complicity of its pathogenesis, low effectiveness of therapy and absence of efficient recommendations in the therapeutic management of this pathology.

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Status indicators of t-cell immunity in hiv-infected persons and patients co-infected with HIV/HCV

Abstract: Features of T-cell immunity have been studied in HIV-infected patients (n=30) and in HIV-infected patients with chronic hepatitis C (n=30). As a control were healthy donors (n=32). In HIV-infected patients found significant decrease in CD4+ and SD45+ T lymphocytes and an increase in the relative and absolute number of CD3+ T lymphocytes. Patients with co-infection HIV/HCV established a significant reduction in the absolute content of CD4+ (p<0,001), CD45+ (p<0,01), and the relative content of CD4+ (p 0,001), CD45+ (p<0,001), as well as the increase in the absolute number of CD3+ (p<0,05) T lymphocytes. Layering HCV for HIV-infection largely worsens the condition of T-cell immunity, causing deep its deficit compensation.

Keywords: HIV-infection, co-infection HIV/HCV, T-cell immunity.

Hepatitis C virus (HCV) and human immunodeficiency virus (HIV) are characterized by their wide distribution and ability to cause health disorders of the working population, thus causing significant morbidity and mortality worldwide. Ukraine - one of the countries of Europe, leads the sad rating of the number of identified HIV positive and AIDS cases and deaths from the disease [1]. Chronic hepatitis C (CHC) is observed in 60-70% of HIV-infected individuals, due to the common modes of transmission of viruses. Co-infection with HIV / HCV is an important public health problem, since viruses, acting synergistically accelerate the progression of liver disease [2]. HIV accelerates the progression of chronic hepatitis C to cirrhosis and hepatocellular carcinoma, thus increases "liver" mortality. Violations of cell-mediated immunity plays a key role in the pathogenesis of HIV infection and have an influence on the strength of the immune system response to specific antigens [3; 4], because these studies focused on the study of the state of T-cell immunity in HIV-infected patients. Thus, insufficient knowledge about the impact of HCV on the performance of T-cell immunity in patients co-infected with HIV / HCV proves the feasibility of their comprehensive study in order to identify their interest in the pathogenesis of this disease.

Materials and methods. Study on the work carried out at the Department of Infectious Diseases of Kharkiv National Medical University, located at the Regional Clinical Hospital of Infectious Diseases of Kharkiv and Kharkiv regional center for prevention and control of AIDS. Features of T-cell immunity were studied in 60 patients: 30 HIV-infected patients and 30 patients co-infected with HIV / HCV. Among the patients surveyed, the number of men were 41 (68.3%), women - 19 (31.7%). Age of patients was 20-63 years. The comparison group consisted of 32 healthy subjects who were matched for age and sex with the patients studied groups.

Patients underwent studies using peripheral blood hematology analyzer ABX PENTRA 60c Plus (HORIBA ABX Diagnostics Inc., France); immunophenotyping using flow cytofluorometry EPICS ™ Xl ™ (Beckman Coulter, USA). Statistical analysis was performed using the software package «Statistica for Windows», 8.0. Methods that were used include: descriptive statistics (numerical description of variables - the arithmetic mean (M), average sampling error (m), definition of the significance of differences (p)), verifying by Student t-test, Fisher's representative samples, the method of correlation of structures [5].

Results. In HIV-infected individuals compared with controls, there is a significant decrease in the relative content of T-helper cells (CD4+) 1.6 times (p<0.001), CD45+ 1.8-fold (p<0.001) and an increase in relative and absolute number of total lymphocytes (CD3+) - 1.1-fold, respectively (p<0.01)